Document Number 1

Entry 1 of 1 File: EPAB Jul 31, 1997

PUB-NO: WO009726913A1
DOCUMENT-IDENTIFIER: WO 9726913 A1
TITLE: A POLYPEPTIDE FROM LUNG EXTRACT WHICH BINDS AMYLOID-beta PEPTIDE
PUBN-DATE: July 31, 1997

INVENTOR-INFORMATION:

NAME	COUNTRY
STERN, DAVID	N/A
SCHMIDT, ANN MARIE	N/A
YAN, SHI DU	N/A

ASSIGNEE-INFORMATION:

NAME	COUNTRY
UNIV COLUMBIA	US

APPL-NO: US09700857
APPL-DATE: January 21, 1997
PRIORITY-DATA: US59207096A (January 26, 1996)

INT-CL (IPC): A61 K 39/395; A61 K 38/00; C07 K 16/00
EUR-CL (EPC): C07K014/705; C07K014/47, C07K016/28

ABSTRACT:

The present invention provides for a method for inhibiting interaction of an amyloid- beta peptide with a receptor for advanced glycation end product on the surface of a cell which comprises contacting the cell with an agent capable of inhibiting interaction of the amyloid- beta peptide with the receptor for advanced glycation end product, the agent being present in an amount effective to inhibit interaction of the amyloid- beta peptide with the receptor for advanced glycation end product on the surface of the cell. Another embodiment of this invention is a method for evaluating the ability of an agent to inhibit binding of an amyloid- beta peptide with a receptor for advanced glycation end product on the surface of a cell which includes: a) contacting the cell with the agent and amyloid- beta peptide; b) determining the amount of amyloid-beta peptide bound to the cell and c) comparing the amount of bound amyloid- beta peptide determined in step b) with the amount determined in the absence of the agent, thus evaluating the ability of the agent to inhibit the binding of amyloid-beta peptide to the receptor for advanced glycation end product on the surface of the cell.

Document Number 4

Entry 4 of 4 File: EPAB Feb 23, 1995

PUB-NO: WO009505604A2
DOCUMENT-IDENTIFIER: WO 9505604 A2
TITLE: METHODS FOR THE DIAGNOSIS OF ALZHEIMER'S DISEASE
PUBN-DATE: February 23, 1995

INVENTOR-INFORMATION:

NAME	COUNTRY
JOHNSON, GINGER	N/A
GHANBARI, HOSSEIN A	N/A
WOLOZIN, BENJAMIN	N/A
MERRIL, CARL R	N/A

ASSIGNEE-INFORMATION:

NAME	COUNTRY
MOLECULAR GERIATRICS CORP	US
US GOVERNMENT	US

APPL-NO: US09408903
APPL-DATE: August 15, 1994
PRIORITY-DATA: US10592293A (August 13, 1993)

INT-CL (IPC): G01 N 33/68; C07 K 16/18; C12 P 21/08; G01 N 27/26
EUR-CL (EPC): G01N027/447; G01N033/68, C07K014/47

ABSTRACT:

A method for the diagnosis of Alzheimer's Disease is disclosed. The method utilizes a unique set of proteins which are found to be altered in concentration in patients with Alzheimer's. The invention also relates to these proteins and their antibodies. Kits which can be used for the diagnostic test are also disclosed.

Document Number 4

Entry 4 of 4 File: EPAB Feb 23, 1995

PUB-NO: WO009505604A2
DOCUMENT-IDENTIFIER: WO 9505604 A2
TITLE: METHODS FOR THE DIAGNOSIS OF ALZHEIMER'S DISEASE
PUBN-DATE: February 23, 1995

INVENTOR-INFORMATION:

NAME	COUNTRY
JOHNSON, GINGER	N/A
GHANBARI, HOSSEIN A	N/A
WOLOZIN, BENJAMIN	N/A
MERRIL, CARL R	N/A

ASSIGNEE-INFORMATION:

NAME	COUNTRY
MOLECULAR GERIATRICS CORP	US
US GOVERNMENT	US

APPL-NO: US09408903
APPL-DATE: August 15, 1994
PRIORITY-DATA: US10592293A (August 13, 1993)

INT-CL (IPC): G01 N 33/68; C07 K 16/18; C12 P 21/08; G01 N 27/26
EUR-CL (EPC): G01N027/447; G01N033/68, C07K014/47

ABSTRACT:

A method for the diagnosis of Alzheimer's Disease is disclosed. The method utilizes a unique set of proteins which are found to be altered in concentration in patients with Alzheimer's. The invention also relates to these proteins and their antibodies. Kits which can be used for the diagnostic test are also disclosed.

Document Number 1

Entry 1 of 2 File: USPT Oct 19, 1999

DOCUMENT-IDENTIFIER: US 5968768 A
TITLE: CD6 ligand encoding sequence

DEPR:
The predicted amino acid sequence of the human homologue of chicken BEN (ALCAM), a type I membrane protein, consists of a 27 amino acid (aa) N-terminal hydrophobic signal peptide, followed by 500 aa extracellular domain, a 24 aa hydrophobic transmembrane domain, and a 32 aa cytoplasmic domain (see FIG. 29). Glycosylation of ALCAM probably accounts for much of the difference between the predicted molecular weight of .about.65 kDa and the 100-105 kDa molecular weight observed by immunoprecipitation (Patel et al, J. Exp. Med. 1994)); Pesando et al, J. Immunol. 137:689 (1986)). Comparison of the aa sequence of ALCAM with others in the data base showed that it was homologous to neurolin (Lawssing et al, Differentiation 56:21 (1994)), a protein expressed by neural axons of the goldfish visual system (38% identity/55% similarity), RAGE (Neeper et al, J. Biol. Chem. 267:14998 (1992)), a receptor for advanced glycation end products (28/43%), and MUC18 (Lehmann et al, Proc. Natl. Acad. Sci. USA 86:9891 (1989)), a cell surface protein whose expression correlates with the metastatic potential of melanoma cells (23/49%) (FIG. 26A).

CCOR:
435/69.1

CCXR:
435/320.1

Document Number 2

Entry 2 of 2 File: USPT Oct 5, 1999

US-PAT-NO: 5962245
DOCUMENT-IDENTIFIER: US 5962245 A

TITLE: Methods for detecting the presence of advanced glycosylation endproducts
DATE-ISSUED: October 5, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Li; Yong Ming	Fresh Meadows	NY	N/A	N/A
Vlassara; Helen	Shelter Island	NY	N/A	N/A
Cerami; Anthony	Shelter Island	NY	N/A	N/A

ASSIGNEE INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY	TYPE CODE
The Picower Institute for Medical Research	Manhasset	NY	N/A	N/A	02

APPL-NO: 8/ 475055
DATE FILED: June 7, 1995
PARENT-CASE:
This Application is a Continuation of application Ser. No. 08/418,642, filed Apr. 7, 1995, now abandoned.

INT-CL: [6] C12 Q 1/34, C12 Q 1/00, G01 N 33/566
US-CL-ISSUED: 435/18; 435/4, 436/501
US-CL-CURRENT: 435/18; 435/4, 436/501
FIELD-OF-SEARCH: 436/501, 435/4, 435/18

REF-CITED:

U.S. PATENT DOCUMENTS

PAT-NO	ISSUE-DATE	PATENTEE-NAME	US-CL
5214028	May 1993	Tomita et al.	514/6

FOREIGN PATENT DOCUMENTS

FOREIGN-PAT-NO	PUBN-DATE	COUNTRY
0474506	March 1992	EP
0629347	December 1994	EP
WO 92/00252	January 1992	WO
WO 93/04086	March 1992	WO
WO 95/20979	August 1995	WO

ART-UNIT: 161
PRIMARY-EXAMINER: Veber; Jon P.
ATTY-AGENT-FIRM: Klauber & Jackson

ABSTRACT:

The present invention is directed to methods for detecting the presence of advanced glycosylation endproducts using the unexpected discovery that certain antibacterial proteins, in particular lysozyme and lactoferrin, bind to advanced glycosylation endproducts (AGEs) with high affinity, and that this binding activity is substantially noncompetitive with binding of bacterial carbohydrates to the antibacterial proteins. Accordingly, the invention relates to diagnostic methods for diseases and disorders associated with increased levels of AGEs, by using compositions having associated therewith a molecule having a hydrophilic loop domain, which domain is associated with AGE-binding activity, and compositions comprising such a domain. The invention further relates to compositions and devices for partitioning AGEs away from a sample.
7 Claims, 18 Drawing figures

Searches for User *jkerr* (Count = 16)

Queries 1 through 16.

S #	Comment	Database	Query
S16		ALL	(435/4.ccls.435/69.1.ccls.435/172.1.ccls.435/368.ccls.435/320.1.ccls.435/455.ccls.) and (advanced adj1 glycation)
S15		ALL	(435/4.ccls.435/69.1.ccls.435/172.1.ccls.435/368.ccls.435/320.1.ccls.435/455.ccls.) and (presenilin-2)
S14		ALL	435/4.ccls.435/69.1.ccls.435/172.1.ccls.435/368.ccls.435/320.1.ccls.435/455.ccls.
S13		ALL	wolozin-benjamin.in.
S12		ALL	yan-shi-du.in.
S11		ALL	(stern-david.in.) and (advanced adj1 glycation)
S10		ALL	(stern-david.in.) and (presenilin-2)
S9		ALL	stern-david.in.
S8		ALL	presenilin-2
S7		ALL	(advanced adj1 glycation) and amyloid
S6		ALL	(advanced adj1 glycation) and (presenilin$1)
S5		ALL	advanced adj1 glycation
S4		ALL	rage and alzheimer$3
S3		ALL	rage
S2		ALL	receptor adj1 for adj1 advanced adj1 glycation
S1		ALL	presenilin$1

Searches for User *jkerr* (Count = 9)

Queries 1 through 9.

S #	Comment	Database	Query
S9		USPT	(800/3.ccls.800/13.ccls.800/14.ccls.536/24.1.ccls.) and ((neutrophil adj1 gelatinase adj1 associated) or (neu-related adj1 lipocalin))
S8		USPT	800/3.ccls.800/13.ccls.800/14.ccls.536/24.1.ccls.
S7		USPT	neutrophil adj1 gelatinase adj1 associated
S6		USPT	neu-related adj1 lipocalin
S5		USPT	transgenic.clm. and gene.clm. and promoter.clm. and mammary.clm.
S4		USPT	transgenic.clm. and gene.clm. and promoter.clm.
S3		USPT	transgenic.clm. and gene.clm.
S2		USPT	transgenic.clm.
S1		USPT	transgenic.clms.

Document Number 1

Entry 1 of 11 File: USPT Dec 7, 1999

DOCUMENT-IDENTIFIER: US 5998172 A
TITLE: Anti-CD6 ligand antibodies

DRPR:
FIGS. 26A to 26C. Sequence analysis of the predicted amino acid sequence of the CD6 ligand designated ALCAM, Northern analysis of ALCAM mRNA expression, and ALCAM cell surface expression by activated T cells. FIG. 26A. Alignment of the immunoglobulin-like extracellular domains of ALCAM (residues 35-512) (residues 35 to 512 of SEQ ID NO:2), BEN (SEQ ID NO:3), neurolin (SEQ ID NO:4), RAGE (SEQ ID NO:5) and MUC18 (SEQ ID NO:6). The lower case letter in front of the protein name designates the species (human, h, chicken, c, and fish, f). Consensus residues are those shared by three or more proteins (SEQ ID NO:7). Invariant residues are shown shaded and Cys's are highlighted with an asterisk. The numbering of the peptide sequences shown, obtained from published manuscripts, are as follows: cBEN, 8-484 (Pourquie et al, Proc. Natl. Acad. Sci. USA 89:5261 (1992)); neurolin, 1-466 (Lawssing et al, Differentiation 56:21 (1994)); RAGE, which contains three Ig domains, 30-307 (Neeper et al, J. Biol. Chem. 267:14998 (1992)); MUC18, 40-525 (Lehmann et al, Proc. Natl. Acad. Sci. USA 86:9891 (1989)). FIG. 26B. 15 .mu.g of total RNA from peripheral blood monocytes and 25 .mu.g of total RNA from resting and PHA activated (72h) peripheral blood mononuclear cells, the T cell lymphomas CEM and MOLT4, the erythroleukemia cell line K562, the B cell lymphomas RAMOS, RAJI and DAUDI, the myelo-monocytic cell lines HL60 and U937, the large granular lymphoma YT, the human breast carcinoma HBL-100 and COS cells were used to prepare an RNA blot. Random primed .sup.32 P-labeled ALCAM or glyceraldehyde 3-phosphate dehydrogenase (GAPDH) cDNA's were used as probes. FIG. 26C. Peripheral blood mononuclear cells were activated in vitro with PHA and the ability of T cells to bind to either CD6-Rg or J4-81 was monitored for a period of ten days by two color immunofluorescence and flow cytometry. The mean channel fluorescence vs. day was plotted.

DEPR:
ALCAM (BEN) in the chicken is expressed predominantly during early embryonic development and in the brain (Pourquie et al, Development 109:743-752 (1990)). ALCAM functions as a homophilic adhesion molecule and supports neurite outgrowth (Tanaka et al, Neuron 7:535-545 (1991), Burns et al, Neuron 7:209-220 (1991)). Expression of human ALCAM by neurons in the brain has been reported (Patel et al, "Identification and

characterization of an 100 kDa ligand for CD6 on human thymic epithelial cells", J. Exp. Med. (1994)). Interactions between the immune and nervous system are important in the pathology of certain chronic neurodegenerative diseases such as multiple sclerosis, Alzheimer's disease, and amyotrophic lateral sclerosis (Appel et al, Advances in Neurology 56:405-412 (1991), McGeer et al, Can. J. Neurol. Sci. 18:376-379 (1991), Rowland, L. P., Advances in Neurology 56:3-23 (1991)). The finding that CD6 and CD6 ligand are both expressed by cells in these systems indicate that this receptor/ligand pair functions in cellular interactions between the immune and nervous systems.

DEPR:
The predicted amino acid sequence of the human homologue of chicken BEN (ALCAM), a type I membrane protein, consists of a 27 amino acid (aa) N-terminal hydrophobic signal peptide, followed by 500 aa extracellular domain, a 24 aa hydrophobic transmembrane domain, and a 32 aa cytoplasmic domain (see FIG. 29). Glycosylation of ALCAM probably accounts for much of the difference between the predicted molecular weight of .about.65 kDa and the 100-105 kDa molecular weight observed by immunoprecipitation (Patel et al, J. Exp. Med. (1994)); Pesando et al, J. Immunol. 137:689 (1986)). Comparison of the aa sequence of ALCAM with others in the data base showed that it was homologous to neurolin (Lawssing et al, Differentiation 56:21 (1994)), a protein expressed by neural axons of the goldfish visual system (38% identity/55% similarity), RAGE (Neeper et al, J. Biol. Chem. 267:14998 (1992)), a receptor for advanced glycation end products (28/43%), and MUC18 (Lehmann et al, Proc. Natl. Acad. Sci. USA 86:9891 (1989)), a cell surface protein whose expression correlates with the metastatic potential of melanoma cells (23/49%) (FIG. 26A).

Document Number 5

Entry 5 of 11 File: USPT Jan 26, 1999

US-PAT-NO: 5864018
DOCUMENT-IDENTIFIER: US 5864018 A

TITLE: Antibodies to advanced glycosylation end-product receptor polypeptides and uses therefor
DATE-ISSUED: January 26, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Morser; Michael John	San Francisco	CA	N/A	N/A
Nagashima; Mariko	Belmont	CA	N/A	N/A

US-CL-CURRENT: 530/387.1; 530/387.3, 530/388.1, 530/388.22, 530/391.3

CLAIMS:

What is claimed is:
1. An isolated monoclonal antibody, wherein said antibody specifically binds to a soluble human receptor to an advanced glycosylation end-product ("RAGE") polypeptide and wherein the polypeptide consists of an amino acid sequence selected from the group consisting of: WKLNTGRTEA (SEQ ID No: 8); CEVPAQPSPQI (SEQ ID No: 9); CRAMNQNGKETKSN (SEQ ID No: 10); GPQDQGTYSC (SEQ ID No: 7); AQNITARIGEPLVLK (SEQ ID No: 12); CKGAPKKPPQ (SEQ ID No: 5); EQTRRHPET (SEQ ID No: 14); RGGDPRPTFSC (SEQ ID No: 15); SPGLPRHRAL (SEQ ID No: 16); and SSHGPQESRA (SEQ ID No: 17).
2. An isolated monoclonal antibody said antibody specifically binds to a soluble human receptor to an advanced glycosylation end-product ("RAGE") polypeptide and wherein the polypeptide consists of an amino acid sequence selected from the group consisting of: WKLNTGRTEAC (SEQ ID No: 6); CKGAPKKPPQ (SEQ ID No: 5); and GPQDQGTYSC (SEQ ID No: 7).
3. The isolated antibody of claim 1, wherein said antibody is humanized.
4. The isolated antibody of claim 2, wherein said antibody is humanized.
5. The isolated antibody of claim 1, wherein said antibody further comprises a labeling group.
6. The isolated antibody of claim 5, wherein said labeling group is selected from the group consisting of a fluorescent label, a radioactive label and a bioactive label.
7. The isolated antibody of claim 2, wherein said antibody further comprises a labeling group.
8. The isolated antibody of claim 7, wherein said labeling group is selected from the group consisting of a fluorescent label, a radioactive label and a bioactive label.

label, a radioactive label and a bioactive label.
9. A composition, comprising an antibody of claim 1 in combination with a pharmaceutically acceptable carrier.
10. A composition, comprising an antibody of claim 2 in combination with a pharmaceutically acceptable carrier.

Document Number 10

Entry 10 of 11 File: DWPI Oct 30, 1998

DERWENT-ACC-NO: 1998-594476
DERWENT-WEEK: 199911

TITLE: Preventing or inhibiting progression of Alzheimer's Disease - comprises use of recombinant DNA encoding an antibody specific for the N- or C-terminus of an amyloid-beta peptide

INVENTOR: CHAIN, D G

PATENT-ASSIGNEE: MINDSET LTD[MINDN], MCINNIS P A[MCINI]

PRIORITY-DATA:

APPL-NO	APPL-DATE
1997US-0041850	April 9, 1997

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 9871034 A	October 30, 1998	N/A	000	A61K048/00
WO 9844955 A1	October 15, 1998	E	058	A61K048/00

DESIGNATED-STATES: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	APPL-DESCRIPTOR
AU 9871034A	April 9, 1998	1998AU-0071034	N/A
AU 9871034A	N/A	WO 9844955	Based on
WO 9844955A1	April 9, 1998	1998WO-US06900	N/A

INT-CL (IPC): A61 K 48/00; C07 H 21/00; C12 N 15/00; C12 P 21/04; C12 P 21/06; C12 P 21/08

ABSTRACTED-PUB-NO: WO 9844955A
BASIC-ABSTRACT:

Prevention or inhibition of progression of Alzheimer's Disease (AD), comprises administering a composition comprising a recombinant DNA molecule containing a gene encoding a

recombinant antibody molecule end-specific for the N-terminus or the C-terminus of an amyloid-beta (AB) peptide (ABP), operably linked to a promoter which is expressed in the central nervous system (CNS), in association with a device for gene delivery, to a patient to prevent the accumulation of ABPs and the aggregation of peptides which form amyloid deposits in the brain. Also claimed are: (1) monoclonal antibody (MAb) end-specific for the N-terminus of an ABP; (2) a recombinant DNA molecule comprising a gene encoding a recombinant antibody molecule end-specific for the N-terminus or the C-terminus of an ABP and a promoter operably linked to the gene, where the promoter is capable of expressing the recombinant antibody molecules in brain cells; (3) a vector comprising a recombinant DNA as in (2), and (4) a host cell transformed with a vector as in (3).

USE - The recombinant antibody molecules prevent the accumulation of ABPs in the extracellular space, interstitial fluid and cerebrospinal fluid and the aggregation of such peptides into amyloid deposits in the brain. They also inhibit the progression of AD by also inhibiting the interaction of ABPs mediating AB induced neurotoxicity and inhibiting the AB induced complement activation and cytokine release involved in the inflammatory process associated with CD and also block the interaction of AB with cell surface receptors such as the RAGE receptor.

ABSTRACTED-PUB-NO: WO 9844955A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/5

DERWENT-CLASS: B04 D16
CPI-CODES: B04-E02A; B04-E08; B04-F0200E; B04-G21; B14-J01B3; D05-H11A; D05-H12A; D05-H12E; D05-H14;

Document Number 11

Entry 11 of 11 File: DWPI Oct 28, 1998

DERWENT-ACC-NO: 1997-526458
DERWENT-WEEK: 199848

TITLE: New soluble advanced glycosylation end-product receptor polypeptide - used for reducing vascular permeability, complications of diabetes etc., also for purification and to screen for modulators

INVENTOR: MORSER, M J; NAGASHIMA, M

PATENT-ASSIGNEE: SCHERING AG[SCHD]

PRIORITY-DATA:

APPL-NO	APPL-DATE
1996US-0633147	April 16, 1996

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
ZA 9703242 A	October 28, 1998	N/A	088	C07K000/00
WO 9739121 A1	October 23, 1997	E	091	C12N015/12
AU 9726960 A	November 7, 1997	N/A	000	C12N015/12

DESIGNATED-STATES: AL AM AT AU AZ BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IL IS JP KE KG KP KR KZ LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG UZ VN AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG

CITED-DOCUMENTS:8.Jnl.Ref; WO 9304086

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	APPL-DESCRIPTOR
ZA 9703242A	April 16, 1997	1997ZA-0003242	N/A
WO 9739121A1	April 11, 1997	1997WO-EP01832	N/A
AU 9726960A	April 11, 1997	1997AU-0026960	N/A
AU 9726960A	N/A	WO 9739121	Based on

INT-CL (IPC): A61 K 0/00; C07 H 0/00; C07 K 0/00; C07 K 1/22; C07 K 14/705; C07 K 16/ 28; C12 N 15/10; C12 N 15/12; G01 N 33/566

ABSTRACTED-PUB-NO: WO 9739121A

BASIC-ABSTRACT:

Pure, biologically active, soluble, human advanced glycosylation end-product receptor (RAGE) polypeptide (I) is new. Also new are: (1) recombinant cells that produce (I) or its active fragments; and (2) nucleic acid (II) encoding (I).

USE - (I), or their mimetics, inhibit interaction between advanced glycosylation end-products (AGE) and a receptor (specifically RAGE), so are used to treat diseases associated with AGE/RAGE interaction, particularly increased vascular permeability, diabetes mellitus (particularly complications such as micro- or macro-vasculopathy or occlusive vascular disorders such as neuropathy, nephropathy, retinopathy or atherosclerosis) or haemodialysis-associated amyloidosis, also (not claimed) activation of microglial cells by beta -amyloid peptides in Alzheimer's disease or age-related disorders such as oxidative stress. (I) are also used, when immobilised, to purify AGE from a protein mixture and to screen for compounds that are (ant)agonists of AGE/RAGE interaction and also (not claimed) diagnosticall y to detect abnormal levels of AGE. Antibodies (Ab) against (I) are useful as immunoassay reagents for measurement of RAGE levels; as inhibitors of interaction between AGE and RAGE or o ther receptors and for purification and quantification of RAGE polypeptides. (II) are used to express recombinant (I) and as probes for isolating (related) genes.

ABSTRACTED-PUB-NO: WO 9739121A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.9A/15

DERWENT-CLASS: B04 D16 S03
CPI-CODES: B04-E01; B04-N04; B14-F02; B14-F07; B14-J01; B14-J01A4; D05-C12; D05-H09; D05-H11A1; D05-H12A; D05-H12D2; D05-H14; D05-H15; D05-H17A4;
EPI-CODES: S03-E14H4;

Document Number 17

Entry 17 of 18 File: EPAB Jul 31, 1997

PUB-NO: WO009726913A1
DOCUMENT-IDENTIFIER: WO 9726913 A1
TITLE: A POLYPEPTIDE FROM LUNG EXTRACT WHICH BINDS AMYLOID-beta PEPTIDE
PUBN-DATE: July 31, 1997

INVENTOR-INFORMATION:

NAME	COUNTRY
STERN, DAVID	N/A
SCHMIDT, ANN MARIE	N/A
YAN, SHI DU	N/A

ASSIGNEE-INFORMATION:

NAME	COUNTRY
UNIV COLUMBIA	US

APPL-NO: US09700857
APPL-DATE: January 21, 1997
PRIORITY-DATA: US59207096A (January 26, 1996)

INT-CL (IPC): A61 K 39/395; A61 K 38/00; C07 K 16/00
EUR-CL (EPC): C07K014/705; C07K014/47, C07K016/28

ABSTRACT:

The present invention provides for a method for inhibiting interaction of an amyloid- beta peptide with a receptor for advanced glycation end product on the surface of a cell which comprises contacting the cell with an agent capable of inhibiting interaction of the amyloid- beta peptide with the receptor for advanced glycation end product, the agent being present in an amount effective to inhibit interaction of the amyloid- beta peptide with the receptor for advanced glycation end product on the surface of the cell. Another embodiment of this invention is a method for evaluating the ability of an agent to inhibit binding of an amyloid- beta peptide with a receptor for advanced glycation end product on the surface of a cell which includes: a) contacting the cell with the agent and amyloid- beta peptide; b) determining the amount of amyloid-beta peptide bound to the cell and c) comparing the amount of bound amyloid- beta peptide determined in step b) with the amount determined in the absence of the agent, thus evaluating the ability of the agent to inhibit the binding of amyloid-beta peptide to the receptor for advanced glycation end product on the surface of the cell.

Document Number 18

Entry 18 of 18 File: DWPI May 3, 1999

DERWENT-ACC-NO: 1999-277439
DERWENT-WEEK: 199937

TITLE: New peptides based on an advanced glyation end product receptor are useful for treating Alzheimer's disease and Down's syndrome

INVENTOR: LAMSTER, I; SCHMIDT, A M ; STERN, D ; YAN, S D

PATENT-ASSIGNEE: UNIV COLUMBIA NEW YORK[UYCO]

PRIORITY-DATA:

APPL-NO	APPL-DATE
1997US-0948131	October 9, 1997

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 9897958 A	May 3, 1999	N/A	000	A61K038/00
WO 9918987 A1	April 22, 1999	E	099	A61K038/00

DESIGNATED-STATES: AU CA JP MX AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	APPL-DESCRIPTOR
AU 9897958A	October 9, 1998	1998AU-0097958	N/A
AU 9897958A	N/A	WO 9918987	Based on
WO 9918987A1	October 9, 1998	1998WO-US21346	N/A

INT-CL (IPC): A61 K 38/00; C07 K 5/00

ABSTRACTED-PUB-NO: WO 9918987A
BASIC-ABSTRACT:

NOVELTY - Novel isolated peptides (I) having an amino acid sequence corresponding to an amino acid sequence of a V-domain of a receptor for an advanced glycation end product (AGEP), are new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a composition comprising (I) linked to an antibody or its portion;

(2) a method for inhibiting an amyloid-beta peptide (ABP) interaction with a receptor for AGEP when the receptor is on the surface of a cell, which comprises contacting the cell with an inhibitor of the interaction to inhibit interaction of the ABP with the receptor for AGEP;

(3) a method for inhibiting degeneration of a neuronal cell which comprises contacting the cell with an inhibitor of the interaction of an ABP with a receptor for AGEP so as to inhibit the interaction and thereby inhibit degeneration of the neuronal cell;

(4) a method for inhibiting formation of an ABP fibril on a cell which comprises contacting the cell with an inhibitor of the interaction of an ABP with a receptor for AGEP so as to inhibit the interaction and thereby inhibit formation of the ABP fibril on a cell;

(5) a method for inhibiting extracellular assembly of an ABP into a fibril which comprises contacting the ABP with an inhibitor of the interaction of an ABP with another ABP so as to inhibit the interaction and thereby inhibit extracellular assembly of an ABP into a fibril;

(6) a method for inhibiting aggregation of ABP on the surface of a cell which comprises contacting the ABP with an inhibitor of the interaction of the ABP with a receptor for AGEP so as to inhibit the interaction and thereby inhibit aggregation of ABP on the surface of a cell;

(7) a method for inhibiting infiltration of a microglial cell into senile plaques which comprises contacting the microglial cell with an inhibitor of the interaction of an ABP with a receptor for AGEP on the surface of the microglial cell, so as to inhibit the interaction and thereby inhibit infiltration of a microglial cell into senile plaques;

(8) a method for inhibiting activation of a microglial cell by an ABP which comprises contacting the microglial cell with an inhibitor of the interaction of the ABP with a receptor for AGEP on the surface of the microglial cell so as to inhibit the interaction and thereby inhibit activation of a microglial cell;

(9) a method for treating a subject with a condition associated with an interaction of an ABP with a receptor for AGEP on a cell, which comprises administering to the subject an inhibitor capable of inhibiting the interaction of the ABP with the receptor for AGEP;

(10) a method for evaluating the ability of an agent to inhibit binding of an ABP with a V-domain of a receptor for AGEP on the surface of a cell which comprises:

(a) contacting the cell with the agent and ABP;

(b) determining the amount of ABP bound to the cell; and

(c) comparing the amount of bound ABP determined in (b) with the amount determined in the absence of the agent, thus evaluating the ability of the agent to inhibit the binding of ABP to the V-domain of the receptor for AGEP on the surface of the cell;

(11) a method for inhibiting activation of a NF-kappaB gene in a cell which comprises contacting the cell with an inhibitor of the interaction of ABP with a receptor for AGEP on the cell so as to inhibit the interaction and thus inhibit activation of NF-kappaB in the cell;

(12) a method for inhibiting periodontal disease in a subject which comprises administering topically to the subject a pharmaceutical composition which comprises soluble receptor for an AGEP (sRAGE) to accelerate wound healing and thereby inhibit periodontal disease;

(13) a method for inhibiting an AGEP's interaction with a receptor for AGEP when the receptor is on the surface of a cell, which comprises contacting the cell with an inhibitor of the interaction to inhibit interaction of the AGEP with the receptor for AGEP;

(14) a method for treating a subject with a condition associated with an interaction of an AGEP with a receptor for AGEP on a cell, which comprises administering to the subject an inhibitor capable of inhibiting the interaction of the AGEP with the receptor for AGEP.

USE - The methods can be used for treating conditions associated with an interaction of an ABP or an AGEP with a receptor for AGEP, e.g. diabetes, Alzheimer's disease, senility, renal failure, hyperlipidemi c atherosclerosis, neuronal cytotoxicity, Down's syndrome, dementia associated with head trauma, amyotrophic lateral sclerosis, multiple sclerosis, amyloidosis, an autoimmune disease, inflammation, a tumor, cancer, male impotence, wound healing, periodontal disease, neuropathy, retinopathy, nephropathy or neuronal degeneration (claimed).

ABSTRACTED-PUB-NO: WO 9918987A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/1

DERWENT-CLASS: B04 D16
CPI-CODES: B04-G01; B14-C03; B14-F06; B14-G02D; B14-H01; B14-J01A4; B14-N03; B14-N06B; B14-N10; B14-N17B; B14-S01; B14-S04; D05-H10; D05-H11;

Document Number 4

Entry 4 of 6 File: EPAB Oct 16, 1997

PUB-NO: WO009738133A1
DOCUMENT-IDENTIFIER: WO 9738133 A1
TITLE: VARIANT PRESENILIN-2 GENES

PUBN-DATE: October 16, 1997

INVENTOR-INFORMATION:

NAME	COUNTRY
HARDY, JOHN	US
GOATE, ALISON M	US
FULDNER, REBECCA A	US

INT-CL (IPC): C12 Q 1/68; C07 H 21/04
EUR-CL (EPC): C12Q001/68; C07K014/47

ABSTRACT:

Variant presenilin-2 genes are provided. Methods of using these genes in diagnosing Alzheimer's disease are also provided.

Document Number 6

Entry 6 of 6 File: DWPI Apr 8, 1998

DERWENT-ACC-NO: 1997-512739
DERWENT-WEEK: 199818

TITLE: Variant presenilin-2 gene - useful for diagnosis of Alzheimer's disease

INVENTOR: FULDNER, R A; GOATE, A M ; HARDY, J

PRIORITY-DATA:
1996US-0014860 April 4, 1996

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 833947 A1	April 8, 1998	E	000	C12Q001/68
WO 9738133 A1	October 16, 1997	E	040	C12Q001/68
AU 9725414 A	October 29, 1997	N/A	000	C12Q001/68

INT-CL (IPC): C07 H 21/04; C12 Q 1/68

ABSTRACTED-PUB-NO: WO 9738133A
BASIC-ABSTRACT:

A variant presenilin-2 (PS-2) gene is claimed.

USE - Alzheimer's disease, particularly in Volga-Germans (a culturally distinct subpopulation in Russia), can be diagnosed using the PS-2 variant sequences or the exonic or intronic sequences of the PS-2 gene. The PS-2 gene variants can also be used in the creation of transgenic animals to be used as disease models (not claimed).
ABSTRACTED-PUB-NO:

WO 9738133A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/2